## REMARKS/ARGUMENTS

Reconsideration of this application is requested. Claims 21-29 are in the case.

## I. SEQUENCE LISTING

In response to the sequence listing requirement, the specification has been amended to include the SEQ ID Numbers for the sequences appearing on pages 8 and 9 of the specification. A paper copy and a computer readable copy of the Sequence Listing are attached. Entry is requested.

## II. SPECIFICATION

An Abstract is presented on a separate sheet, in accordance with the requirement appearing on page 3 of the Action. In addition, a new title is presented.

## III. THE 35 U.S.C. § 112, SECOND PARAGRAPH, REJECTION

Claims 1-20 stand rejected under 35 U.S.C. § 112, second paragraph, as allegedly indefinite for the reasons detailed beginning on page 3 of the Action. That rejection is respectfully traversed.

The claims in the present application have been cancelled without prejudice, and replaced by new claims 21-29. The Examiner's formal points have been taken into consideration in preparing the new claims. The following comments are offered.

The Examiner asserts that the claims do not clearly define the invention described in the specification. In particular, claim 1 has been rejected as failing to adequately define the "compound", the "structural tegument protein", the "neurotropic

virus" and the "motor protein". In addition, the Examiner contends that several of the claims are vague and indefinite due to the recitation of certain allegedly relative terms and phrases, e.g., "reducing", "capable of altering" and "motor protein-like".

The new claims presented herewith are believed to obviate the outstanding formal points. The amended claims clearly define the invention disclosed in the specification. In this regard, the Examiner's attention is drawn to the restriction of the claims to **Herpes simplex virus (HSV)** and the requirement that the "compound" **specifically** prevents binding between the **US11** structural tegument protein of HSV and the **kinesin** motor protein of the neuron or cell. The word "reducing" not longer appears in the claims.

With regard to the Examiner's objection to the term "mimic", terms such as "a mimic of kinesin" and "mimic of US11" would be clear to a person skilled in this art. The mimics are defined in the claims by way of a functional requirement (i.e., to specifically prevent binding between US11 and kinesin).

In response to the Examiner's comments in the first full paragraph on page 4 of the Office Action, new claim 21 adequately defines a method. The additional steps identified by the Examiner, such as "when to add, where to administer, how much, what to detect, when to detect and how to monitor the "prevention" of the virus transport" are clearly not essential features of that method.

Withdrawal of the outstanding 35 U.S.C. § 112, second paragraph, rejection would now appear to be in order. Such action is respectfully requested.

## IV. THE 35 U.S.C. § 112, FIRST PARAGRAPH, REJECTIONS

Claims 1-20 stand rejected under 35 U.S.C. § 112, first paragraph, on alleged lack of enablement grounds. In particular, the Examiner asserts that the claims are not fully enabled and, at pages 10 to 12, the Examiner additionally asserts that the specification fails to describe the claimed subject matter in such a way as to reasonably convey to a person skilled in the art that the inventors, at the time the application was filed, had possession of the claimed invention. The rejections are respectfully traversed.

The specification is clearly enabling with respect to the invention as claimed, and also clearly establishes that when, the application was filed, the inventors were in possession of the invention. For example, page 4 lines 7 to 22 of the specification details suitable compounds that can prevent binding between US11 and kinesin. In addition, and as acknowledged by the Examiner, the specification enables an *in vitro* method of screening compounds that inhibit the transport of HSV in a neuron or cell through preventing binding between US11 and kinesin. Clearly, therefore, a person skilled in the art, having read the present specification, would have been able to routinely identify and provide further compounds able to prevent transport of HSV within a neuron or cell through preventing binding between US11 and kinesin.

Based upon the support provided in the specification, and in view of the Examiner's comments, new claims 27-29 are presented directed to a method of screening for compounds that inhibit the transport of HSV in a neuron or cell through preventing binding between US11 and kinesin. No new matter is entered.

In light of the above, it is clear that the invention as claimed is fully enabled by the specification as originally filed, and is described in such a way as to reasonably convey to a person skilled in the art that the inventors, at the time the application was filed, had possession of the claimed invention.. Reconsideration and withdrawal of the outstanding lack of enablement rejections are accordingly respectfully requested.

## V. THE ANTICIPATION REJECTIONS

Claims 12-20 stand rejected under 35 U.S.C. § 102(b) as allegedly anticipated by Roller et al. The Examiner alleges that the cited document discloses an antibody that would bind to the US11 tegument protein of HSV virus and would therefore inhibit its function. The rejection is respectfully traversed.

Contrary to the Examiner's assertion, the anti-US11 monoclonal antibody disclosed by Roller et al (1992) would **not** inhibit binding between US11 and the motor protein, kinesin. That is, the anti-US11 monoclonal antibody has been subsequently shown by Roller et al (1996 70:2842-2851) and Diefenbach et al 2002 (J Virol. 2002, 76:3282-3291; copy attached) to bind to an epitope in the amino terminus of US11 (specifically, amino acids 5-35), which is remote from the C-terminus site of interaction with kinesin (see Diefenbach et al 2002). Therefore, the monoclonal antibody of Roller et al (1992) would not inhibit binding between US11 and kinesin. In addition, the disclosed anti-US11 monoclonal antibody would not be able to access the interior of a neuron or cell to inhibit the binding between US11 and kinesin.

Withdrawal of the outstanding anticipation rejection based on Roller et al is believed to be in order. Such action is respectfully requested.

Claims 12-20 stand rejected under 35 U.S.C. § 102(b) as allegedly anticipated by Sodeik et al. That rejection is respectfully traversed.

Sodeik describes compounds that depolymerise micro-tubules and reduce infection of HSV by colchicine and the toxic agents vinblastine and nocodazole (see, in particular, page 1016, right column, last paragraph). In contrast, the compounds referred to in the amended claims are required to specifically prevent binding between US11 and kinesin. Therefore, the mechanism of action of the compounds of the cited document is distinct to the compounds of the present invention.

In light of the above, it is clear that the outstanding anticipation rejection based on Sodeik et al should now be withdrawn. Such action is respectfully requested.

Claims 1, 2, 4-6, 8, 9, 10 and 11 stand rejected under 35 U.S.C. § 102(e) as allegedly anticipated by U.S. Patent 6,326,402 to Kun et al. That rejection is respectfully traversed.

Kun allegedly describes a method of treating a viral infection such as a HSV infection with a compound that "would inhibit the binding of micro-tubules". Kun discloses compounds named diiodo thyronine analogues which bind micro-tubules and inhibit their formation or polymerisation. Therefore, this mechanism of action is also distinct from that of the present invention, wherein the compound is required to specifically prevent binding between US11 and kinesin.

Moreover, it is to be noted that compounds disclosed by Sodeik and Kun can be said to act in a non-selective manner to disrupt the function of human cells by inhibiting the transport of cellular proteins and organelles within the cells. In contrast, the invention of the amended claims requires that the compound specifically prevents

binding between US11 and kinesin and, thereby, does not disrupt other cellular protein interactions in the cell.

Claims 12-20 stand rejected under 35 U.S.C. § 102(e) as allegedly anticipated by three patents to Beraud et al (namely, U.S. Patent 6,346,410, 6,455,293 and 6,492,158). Those rejections are respectfully traversed.

The Examiner alleges that the cited Beraud patents describe compounds that "would bind kinesin and as a consequence would inhibit the infection of HSV". In response, the Beraud do not disclose compounds which bind to kinesin that binds to US11. That is, the Beraud patents disclose compounds which bind to the kinesins, KIF6 and KIF21. These kinesins are distinct from KIF5B which is now known to bind to US11. It is also to be noted that US11 interacts with the cargo-binding domain of KIF5B (see Diefenbach et al, 2002), which lies outside of the motor domain conserved across kinesin proteins. Therefore, compounds directed to the conserved kinesin motor domain would not inhibit binding between US11 and kinesin so as to prevent transport of HSV within a neuron or cell.

Withdrawal of the outstanding anticipation rejections based on the Beraud patents is now believed to be in order. Such action is respectfully requested.

## VI. DRAWINGS

The informalities of the drawings have been noted. Corrected formal drawings are attached.

Allowance of the application is awaited.

Respectfully submitted,

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Attachments: Sequence Listing Paper Copy and Computer Readable Copy; IDS and IDS fee; extension request; corrected drawings